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# Isolated exertional dyspnea as the only symptom in patients with chronic thromboembolic pulmonary hypertension post-pulmonary embolism

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## Abstract

Dyspnea, is a common symptom encountered in primary care settings. While it is frequently associated with existing comorbidities, there are rare conditions that may manifest solely as dyspnea during physical activity. One such condition is chronic pulmonary embolism, which can lead to the development of chronic thromboembolic pulmonary hypertension (CTEPH) (B. Ganatra, R., 2023). Primary care physicians must consider these rare but significant etiologies of exertional dyspnea when there is a lack of clinical improvement following the initial evaluation and management.

Dyspnea is often linked to cardiac and respiratory diseases, but it can also be caused by obesity and deconditioning. Population-based studies have shown a prevalence of 9 to 13% for mild to moderate dyspnea among community-residing adults, and up to 37% of adults aged 70 years and older. (Parshall et al., 2012). In the United States, dyspnea accounts for 3 to 4 million emergency department visits annually (Parshall et al., 2012).

**Keywords:** chronic thromboembolic pulmonary hypertension, pulmonary embolism, dyspnea, exertion, pulmonary hypertension, echocardiogram.

# Introduction

Pulmonary embolism is a common medical condition that often occurs alongside various significant comorbidities. A particularly alarming complication that can arise after experiencing a pulmonary embolism is chronic thromboembolic pulmonary hypertension (CTEPH). This condition poses serious health risks, resulting in considerable functional limitations and increased mortality rates (Paolo Prandoni et al., 2022). Individuals who suffer from pulmonary embolism may also experience post-PE syndrome, which adversely impacts pulmonary circulation, gas exchange, and cardiac performance, manifesting in symptoms such as shortness of breath and diminished physical endurance. Research indicates that not all patients with pulmonary embolism achieve complete thrombus resolution, even with adequate anticoagulation therapy. This incomplete resolution can result in right ventricular dilation and pulmonary hypertension. In a clinical trial, it was found that 44% of patients exhibited at least one echocardiographic indicator of pulmonary hypertension or right ventricular dysfunction after three years of anticoagulation treatment (Nilsson, L et al., 2021).

#### **Case Presentation**

A 59-year-old woman presented to the primary care clinic with a two-year history of dyspnea on exertion, which has progressively worsened during physical activity. She denied experiencing any chest pain or lower extremity swelling and was uncertain about the presence of wheezing, reporting no additional symptoms. The patient's weight has been stable for the last two years.

Her medical history included mild chronic obstructive pulmonary disease (COPD), for which she was prescribed Umeclidinium bromide inhaler to be used once daily. She also had a diagnosis of hypertension managed with Candesartan at a dosage of 8 mg daily, and dyslipidemia treated with Atorvastatin at 40 mg per day. Notably, she had a pulmonary embolism nearly two years prior, which occurred in conjunction with femoral fracture surgery; since that event, she has been on Rivaroxaban at a dosage of 20 mg. The patient has a smoking history of 20 pack-years but quit smoking five years ago.

During the physical examination, the patient presented as alert and oriented. Her vital signs included a blood pressure of 128/72 mmHg, an oxygen saturation of 93% on room air, a temperature of 36.2 °C, a heart rate of 78 beats per minute (regular), and a respiratory rate of 14 breaths per minute. The cardiovascular assessment revealed normal heart sounds without any murmurs, the jugular venous pressure was not observable, and there was no edema in the lower

extremities. The respiratory evaluation was unremarkable, demonstrating normal and symmetrical air entry bilaterally, with no evidence of crackles or wheezing.

A blood examination and a chest X-ray were ordered. The laboratory findings revealed the following values: Red Blood Count at 6.0 x 10^12/L, Hemoglobin at 125 g/L, Platelet Count at 166 x 10^9/L, White Blood Count at 4.6 x 10^9/L, estimated Glomerular Filtration Rate (eGFR) at 74 ml/min, Sodium (Na+) at 140 umol/L, Potassium (K+) at 4.0 umol/L, and D-Dimer at 2850. At the time the patient was contacted and referred to the hospital, the results of the chest X-ray were still pending.

6.0 10^12/L
125 g/L
166 10^9/L
4.6 10^9/L
74 ml/min
140 umol/L
4.0 umol/L
2850

## **Blood Test Results**

In the hospital, a computed tomography pulmonary angiography (CTPA) was conducted, revealing several regions of calcified pulmonary emboli, dilation of the pulmonary arteries, and hypertrophy of the right ventricle. These CTPA findings culminated in a diagnosis of chronic pulmonary embolism (PE) and chronic thromboembolic pulmonary hypertension (CTEPH), which have been responsible for the persistent exertional dyspnea experienced by the patient. The cardiology team engaged in discussions with the patient regarding potential treatment options, including pulmonary endarterectomy, pulmonary balloon angioplasty, and the initiation of epoprostenol therapy.

### **Discussion and Conclusion**

The patient discussed in this case exhibited a Well's score of 1.5, categorizing her as low risk for pulmonary embolism (PE). This assessment is

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further supported by her adherence to lifelong anticoagulation therapy. Diagnostic evaluations revealed that the prior PE had not fully resolved, even with ongoing anticoagulation treatment, resulting in chronic thromboembolic pulmonary hypertension (CTEPH). This condition has been identified as the underlying cause of the patient's exertional dyspnea.

Unexplained dyspnea frequently occurs in primary care settings. Individuals with a history of pulmonary embolism (PE), even when receiving appropriate daily anticoagulation therapy, may still experience residual and unresolved thrombosis. This condition can progressively result in pulmonary hypertension and right ventricular enlargement, contributing to exertional dyspnea and potentially resulting in severe complications (Nishiyama, K.H. et al., 2018).

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